Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION

50 WEST 50TH STREET, NEW YORK, N. Y.

DR. JAMES G. CARR, Chicago, *Editor*

DR. GILBERT H. MARQUARDT, Chicago, *Associate Editor*

Vol. IX August, 1940 No. 8

TACHYCARDIA AND ITS TREATMENT

Introduction

Rapid action of the heart is the most common and obvious cardiac manifestation and therefore, historically, it was the first to be recognized, both by the subject himself and by observers. In fact, palpitation due to tachycardia and arrhythmia was the only symptom ascribed to the heart until some 200 years ago when gradually dyspnea and finally angina pectoris began to be associated with heart disease. The varieties and causes of tachycardia are so numerous that it is necessary first to present a classification.

Classification of Tachycardia

The most common type of tachycardia is that originating in the sinoauricular node. This is called sinoauricular tachycardia, or, for short, sinus tachycardia. Next in frequency is auricular paroxysmal tachycardia, followed by the rapid heart action secondary to fibrillation and flutter of the auricles. A relatively rare form of tachycardia, but much more important, is that arising in the ventricles, which in turn may evolve into ventricular flutter and fibrillation. The rarest paroxysmal tachycardia is that originating in the a-v node.

Sinoauricular Tachycardia

It is largely a matter of opinion at what heart rate sinoauricular tachycardia may be considered present. It seems reasonable to label rates of over 100 in adults with sinus rhythm as sinoauricular tachycardia, over 120 in children, and over 150 in infants. Although there is a wide range of the normal pulse rate at rest, it is rare to find in perfectly normal persons resting heart rates of 100 or more. One always must suspect some underlying exciting factor for such a rate.

The **mechanism** of sinus tachycardia is two-fold, consisting of (1) the removal in whole or in part of parasympathetic (vagus) inhibition of the sinoauricular nodal pacemaker, and (2) stimulation of the sinoauricular nodal pacemaker by an increase in sympathetic nerve excitation. Usually both factors operate more or less simultaneously.

The various causes of normal sinoauricular tachycardia are commonly effort, excitement, and the ingestion of food occurring in ordinary daily life in all individuals, healthy and diseased. A rare type of normal sinoauricular tachycardia is that voluntarily induced, associated largely with sympathetic nerve control. It has been shown that the person who can voluntarily accelerate his heart beat increases his blood pressure and dilates his pupils at the same time. Pathological causes of sinoauricular tachycardia include particularly infections, the pulse rate varying with the temperature; in general one degree of fever is attended by a rise of about ten pulse beats per minute. A second cause consists of a variety of non-infectious toxic states. A prominent one of these is the so-called thyrotoxicosis secondary to hyperthyroidism (exophthalmic goiter). Another pathological cause is infarction of some part of the body in a large enough area to give rise to reactions with or without fever. A state of shock may or may not be associated with tachycardia. Sometimes the cause of sinoauricular tachycardia cannot be discovered.

The **diagnosis** of sinoauricular tachycardia is generally obvious and clearly related to the exciting factor. In rare cases electrocardiographic help is needed. The heart rhythm is usually regular but may wax and wane a little with forced respiration. The rates range from 100 to 200 or somewhat more in children and infants.

The **prognosis** of sinoauricular tachycardia in itself is good. The prognosis in any given case depends entirely on the underlying cause of the tachycardia, except in those instances where serious heart disease is already present and the myocardial or coronary reserve clearly limited, when the tachycardia in itself may precipitate heart failure.

The **treatment** of sinoauricular tachycardia is that of the underlying disease or other factor. Much medicine has been wasted in the past in the effort to reduce the heart rate in sinus tachycardia secondary to infection or other pathological cause. Such efforts are almost invariably unavailing. There are rare exceptions when in the presence of heart disease it may be important to avert failure. Digitalis then may help to maintain myocardial tone, and also in slight degree may even reduce the sinus rate. One must, however, be careful to avoid excessive digitalization and to recognize that digitalis itself may be a cause of ectopic tachycardias. Quinidine is ineffective in sinoauricular tachycardia. Sedatives such as bromides may help a little when there is a large nervous element responsible.

Auricular Paroxysmal Tachycardia

This is the most common of the abnormal or ectopic tachycardias. Its incidence is impossible to determine with any accuracy because many normal persons as well as patients have short paroxysms of tachycardia lasting a few seconds or a few minutes that are not interpreted as such either because they are not sufficiently troublesome to excite concern on the part of the person affected or because there is too little opportunity to obtain a record by electrocardiogram or mechanical pulse tracing owing to the brevity of the paroxysms. Hence statistics from any electrocardiographic laboratory in this respect are grossly inadequate. It seems likely that at least one hundred times as many paroxysms of tachycardia occur through the community at large as can ever be recorded graphically.

The **mechanism** of auricular paroxysmal tachycardia is not certain. There is a rapid and usually

regular sequence of abnormal heart beats originating in the auricle, evidently either from a given point outside the normal pacemaker or as a type of circus wave reentering auricular muscle which in turn has recovered rapidly from its refractory stage. Apparently any part of the auricular musculature may be the site of origin of a paroxysm of auricular tachycardia. Electrocardiographically the P waves of the paroxysm are abnormal in shape, poorly marked in Lead 1 as a rule, often upright in Lead 2 but of not the usual shape, and sometimes inverted. In Lead 3 the P waves may be of varied shape, more closely resembling those in Lead 2 than those in Lead 1, but it must be remembered that normally the P waves in Lead 3 may be diphasic or even inverted. Hence Lead 2 is as a rule the most satisfactory lead for the study of the shape of the P waves in ectopic tachycardias. Lead 4, the routine chest lead, shows the P waves too poorly marked for satisfactory analysis, but a special chest lead may be taken with the exploring electrode over the sternum or just to the right of the sternum in about the 4th space; such a lead may give a clear picture of the auricular action.

Etiology. Auricular paroxysmal tachycardia occurs at all ages, but not so commonly in children as in adults. It is more common in infants than has been previously suspected, however. Dr. John Hubbard* of Boston has encountered five cases in a single year in infants under one year of age, where previously only 19 undoubted cases of paroxysmal auricular tachycardia or flutter in such young babies were on record in the literature.

The exciting factors of auricular paroxysmal tachycardia are legion. It would be impossible to name them all. The most common are effort, excitement, excessive tobacco, thyrotoxicosis, and heart disease itself, especially mitral stenosis. The majority of persons suffering from paroxysmal tachycardia are, however, perfectly normal in health otherwise and with normal hearts. Only a small minority actually have heart disease and yet if we compare the relative incidence in normal persons and in cardiac patients we find a considerably higher incidence in the cardiac patients.

The **diagnosis** of auricular paroxysmal tachycardia may be made usually with ease, without recourse to the electrocardiograph. The sudden onset and usually sudden offset with an approximate doubling of heart rate, and duration of a few minutes to a few hours mark the attack as of this type. Sometimes the attack does not seem to stop abruptly, due to the fact that although the rate drops considerably at the end of the paroxysm the sinoauricular rate when the heart resumes normal rhythm is elevated by excitement or otherwise, preventing the same sensation of marked change in heart rate that occurred at the onset of the attack; for example at the beginning of a paroxysm of tachycardia the rate may jump from 70 to 160, but at its cessation the rate may drop abruptly from 160 only to 120 when the sinus rhythm is restored, and then gradually the sinus rate recedes to normal. It is helpful in doubtful cases to obtain an electrocardiogram during the attack, especially if it is prolonged. The electrocardiogram is almost invariably diagnostic but there are a few puzzling records in which the electrocardiogram is not adequate (except in a record of the onset) in distinguishing paroxysmal auricular tachycardia from sinus tachycardia or from auricular flutter, and in a few instances also bundle-branch block may complicate auricular paroxysmal tachycardia so that there is an imitation of ventricular paroxysmal tachycardia.

Prognosis. The prognosis of auricular paroxysmal tachycardia is generally excellent as to life, but there are instances of such excessive tachycardia that even in a normal person congestive failure, particularly involving the right ventricle, may ensue with distress and even danger; this is especially true in infants when the heart rate may rise to 300 or more. Another exception to the rule that paroxysmal tachycardia is unimportant (though it may be disagreeable) is in the presence of serious heart disease. There are three cardiac conditions which are especially prone to be overburdened by tachycardia: (1) mitral stenosis in which case the pulmonary circulation is flooded with blood, and marked dyspnea, and even pulmonary edema and cardiac asthma, may result; (2) any case of strain and enlargement of the left ventricle (commonly the result either of hypertension, aortic valve disease, or myocardial infarction) in which case the tachycardia may induce left ventricular failure and pulmonary edema; and (3) coronary insufficiency which cannot stand the strain of the tachycardia, a status anginosus then resulting, which in itself is very disagreeable and hazardous and simulates coronary thrombosis.

The prognosis as to recurrence of attacks varies greatly, from but one attack in a long lifetime to paroxysms almost every day, week, or month for many years.

The **treatment** of auricular paroxysmal tachycardia is generally simple enough, since *reassurance*, rest at the time of the paroxysms, and the omission of the exciting factors usually suffice. Most paroxysms are so brief that they need no particular therapy. Sometimes a simple change in position will stop the attack, as for example, stooping over or lying down. One very simple and occasionally helpful method of stopping an attack is firm pressure for a few seconds on the carotid sinus, more effectively on the right side of the neck than on the left. Perhaps one in twenty cases gets immediate relief from this manoeuver. If, however, the paroxysm is prolonged or very disagreeable, other therapy may be needed, both in treatment and in prevention.

The most commonly used and effective of the drugs in the treatment of an attack is quinidine sulphate in the dose of 3 to 6 grains to be repeated once or twice or even three times at two hour intervals, and to be used in a prophylactic way in a dosage of 3 grains every few hours for a day or two at a time or longer if needed. In emergency quinidine or quinine may be given parenterally, most readily in the form of quinine dihydrochloride in the dosage of 7½ grains intramuscularly at two hour intervals if the attack is a severe one and relief must be obtained; it is probably even more effective for ventricular than for auricular paroxysmal tachycardia. Another method of using the cinchona preparations is that proposed by Hepburn and Rykert* who advised for ventricular paroxysmal tachycardia intravenous injection by the drip method of a 10 per cent solution of quinidine sulphate; 50 to 60 grains can be dissolved by vigorous shaking in 500 c.c. of 5% glucose solution or in normal saline. The solution is then filtered, slightly warmed, and given at the rate of 100 to 120 c.c. per hour until normal rhythm results or toxic symptoms (of cinchonism) appear.

Digitalis has not been advised in recent years in the routine treatment of auricular paroxysmal tachycardia although it formerly was used widely. It was found so often ineffective and so often in itself irritating that the pendulum swung too far. It now and then can be distinctly helpful and even better than quinidine or quinine, both in preventing and in treating paroxysms. Digitalization may be fairly

*Reported at meeting of the New England Heart Association at the Children's Hospital, Boston, February, 1940.

*Hepburn, J., and Rykert, H.E.—American Heart Journal 1937, XIV, 620.

rapidly effective, but massive doses are unnecessary; probably three grains or 2 cat units of the leaf every two hours for four or five doses should suffice if the attack lasts that long. Digitalization may be maintained thereafter if the attacks are common and if this drug seems to control them. In babies with paroxysmal tachycardia Dr. Hubbard* has found digitalis apparently invaluable and has given up to 2 or 3 grains in divided doses in the course of 24 to 48 hours.

Other preparations are less important. Potassium salts have had a vogue and in rare cases may have some effect but they have usually been disappointing. It is advisable to withhold morphine because of its unnecessarily strong effects in other directions and because of the possibility of habit formation since paroxysms are often repeated.

There are, however, two other vagotonic drugs that are useful in very resistant cases. The first is syrup of ipecac which acts through the production of marked increase in vagal tone. Two to four drachms (8-16 c.c.) of the syrup at a dose to be repeated in a few hours or up to the point of vigorous vomiting have stopped obstinate attacks. Mecholyl (acetyl-beta-methylcholine chloride) is the other drug which in the dosage of 20 to 50 mgms. injected subcutaneously can stop obstinate attacks, but this drug itself has disagreeable side reactions and should be used with caution, atropine (1/60th grain or 1 mgm. of the sulphate) being at hand as an antidote in case of need.

Auricular Flutter

Auricular flutter is relatively uncommon but it may explain a few of the paroxysms of tachycardia that occur in fairly normal individuals which are never recorded graphically. In an electrocardiographic laboratory series it is one of the rarer disorders of rhythm.

Mechanism. Auricular flutter is apparently the result of a regular circus action in the auricle in the neighborhood of the sinus node. The usual rate of the auricle in flutter is between 200 and 300 and the ventricular rate half that, due to the occurrence of two to one block; rarely one to one rhythm exists.

Etiology. The condition occurs in adults chiefly, in both sexes, and the exciting factors are the same as for auricular paroxysmal tachycardia except that there is a much higher incidence of heart disease, that is, the majority of the patients who show auricular flutter have heart disease, particularly mitral stenosis.

The **diagnosis** is easily made by electrocardiogram and with difficulty in any other way. Temporary halving of the heart rate by carotid sinus pressure is, however, a clue; the 2:1 a-v block is thereby changed to 4:1 pro tem. Attacks are generally more prolonged than are those of simple auricular paroxysmal tachycardia.

The **prognosis** is less favorable than for auricular paroxysmal tachycardia because of the higher incidence of heart disease in the group, and because of the longer paroxysms. Sometimes the flutter continues in more permanent form lasting for years.

The **treatment** is simpler than that for auricular paroxysmal tachycardia for the drug par excellence is digitalis. Digitalization should be maintained either until the attack is over, with the re-establishment of normal rhythm, unless the state of the heart demands the maintenance of digitalization because of myocardial weakness, or permanently if there ensues persistent auricular fibrillation. Quinidine sulphate will restore normal rhythm in some cases, but it is less useful in the long run than is digitalis. Carotid sinus pressure is ineffective; it simply increases temporarily the grade of block. The other drugs mentioned for the treatment of auricular paroxysmal tachycardia are not indicated.

*Personal communication.

Auricular Fibrillation

Auricular fibrillation, much commoner than auricular flutter in the ratio of about fifteen to one, is infrequently found in the absence of heart disease. Nevertheless there is a considerable number of normal persons who have paroxysms of auricular fibrillation. I have personally seen at least 100 such and follow up studies have indicated that there may be neither heart disease nor deleterious effects from the paroxysms of auricular fibrillation. Attacks are often very infrequent and may actually occur only once.

The **mechanism** of auricular fibrillation is like that of flutter but with a circus wave at a faster and irregular rate and irregular ventricular response. The heart rate in a paroxysm of auricular fibrillation is usually close to 150 and absolutely irregular.

Etiology. Both sexes and all ages after early childhood are affected by paroxysms of auricular fibrillation. However, such attacks increase in frequency with increasing years and are common in old age. The most prominent underlying factors are mitral stenosis, thyrotoxicosis, and a combination of hypertension and coronary disease. Excitement, effort, tobacco, infection, and infarction are the most common exciting factors.

Diagnosis. The electrocardiogram gives immediate evidence of the condition and is of great diagnostic aid when there is a question as to clinical interpretation. It may be said, however, that any tachycardia at a rate of over 120 that is grossly irregular is almost certainly the result of auricular fibrillation.

The **prognosis** in auricular fibrillation depends on two factors, (1) the underlying heart condition and (2) the treatment. In the absence of heart disease the prognosis is excellent and the attack usually subsides spontaneously and may never recur. In the presence of heart disease the prognosis depends on the severity of the heart disease and also somewhat on the ease with which the ventricular rate can be controlled by treatment. It is possible to have recurrent auricular fibrillation over periods of many years, as long as twenty or twenty-five or more; on the other hand, if the heart is in very serious condition the onset of auricular fibrillation may kill the patient within a few hours.

The **treatment** of auricular fibrillation is much like that just outlined for auricular flutter. Ordinarily, digitalization is wisest, but for a paroxysm that has recently come on, that is, within a few hours or days, the use of quinidine sulphate may be the best measure. The patient should be at rest and take six grains of quinidine sulphate every two hours for three to six doses as needed, under close observation and preferably with electrocardiographic control. The record should be taken every two hours or just before the next dose is due in order to observe the changing rate of the auricular circus which generally slows as the case evolves favorably, along with an increasing ventricular rate; it is also important to watch electrocardiographically for toxic effects, particularly the occurrence of bundle-branch block. The other measures noted under auricular paroxysmal tachycardia are ineffective or harmful in the presence of auricular fibrillation. Morphine is usually unnecessary and to be avoided unless there is acute pulmonary edema or the status anginosus, as described above under the section on auricular paroxysmal tachycardia. Often paroxysms stop spontaneously in a few hours.

A-V Nodal Paroxysmal Tachycardia

This is a very rare and unimportant variety of paroxysmal tachycardia to be diagnosed only by electrocardiogram and then only with great caution. It is due to rapid impulse formation in the a-v node of Tawara with control of both ventricles and auricles from that point (the P waves of the electrocardiogram are inverted and just follow, precede, or occur simultaneously with the QRS waves). It is to be considered in all its aspects and to be treated like auricular paroxysmal tachycardia.

Ventricular Paroxysmal Tachycardia

Ventricular paroxysmal tachycardia is much less common than auricular paroxysmal tachycardia in the ratio of about one to six.

The **mechanism** is doubtless similar to that in auricular paroxysmal tachycardia or auricular flutter, except that the impulse originates in the ventricular muscle or bundle branches.

Etiology. It occurs in both sexes but is much more limited to older persons than is auricular paroxysmal tachycardia; it is rare in youth. It is a much more serious condition because of the higher incidence of important heart disease or toxic states. Exciting factors are as noted above under auricular paroxysmal tachycardia, but the underlying factors are mainly myocardial infarction, usually fairly fresh, and digitalis poisoning of high degree. Rarely does it occur in normal individuals, but we have seen a few young persons with apparently normal hearts who have shown ventricular paroxysmal tachycardia rather than auricular paroxysmal tachycardia.

The **diagnosis** is to be made with certainty only by electrocardiogram and is shown by the abnormally shaped QRS waves which resemble repeated ventricular premature beats, the heart rate usually running at about 160. The auricles beat independently and the P waves may sometimes be clearly seen superimposed on the QRS and T waves. It is to be distinguished from auricular paroxysmal tachycardia with bundle-branch block, but this it is not always possible to do, even by electrocardiogram, unless the independent P waves are clearly identified. Clinically, one may suspect ventricular paroxysmal tachycardia in contrast to auricular paroxysmal tachycardia if there is a definite but slight arrhythmia or anisosphygmia during the paroxysm. The duration of the attacks is much like that of auricular paroxysmal tachycardia, that is, minutes or hours, rarely days or weeks, and almost never prolonged beyond that time.

The **prognosis** is unfavorable in general because of the severe underlying heart disease or toxic state and because of the ominous experiences of the past. As I have said above there are few normal cases who have had such paroxysms and a number who with heart disease really do well, but for most cases ventricular paroxysmal tachycardia presages a short life, sometimes only a few hours, and rarely more than a few months or years, unless some toxic condition like digitalis poisoning can be blamed and subsides quickly.

The **treatment** of ventricular paroxysmal tachycardia is like that of auricular paroxysmal tachycardia especially with respect to the use of quinidine; this drug is in fact more effective in ventricular than in auricular paroxysmal tachycardia. The other preparations have not seemed to be effective or advisable. Absolute rest is essential and morphine is more often indicated in emergency than in the other conditions noted above, in which it should almost always be withheld.

Paul D. White, M.D.
Boston, Mass.